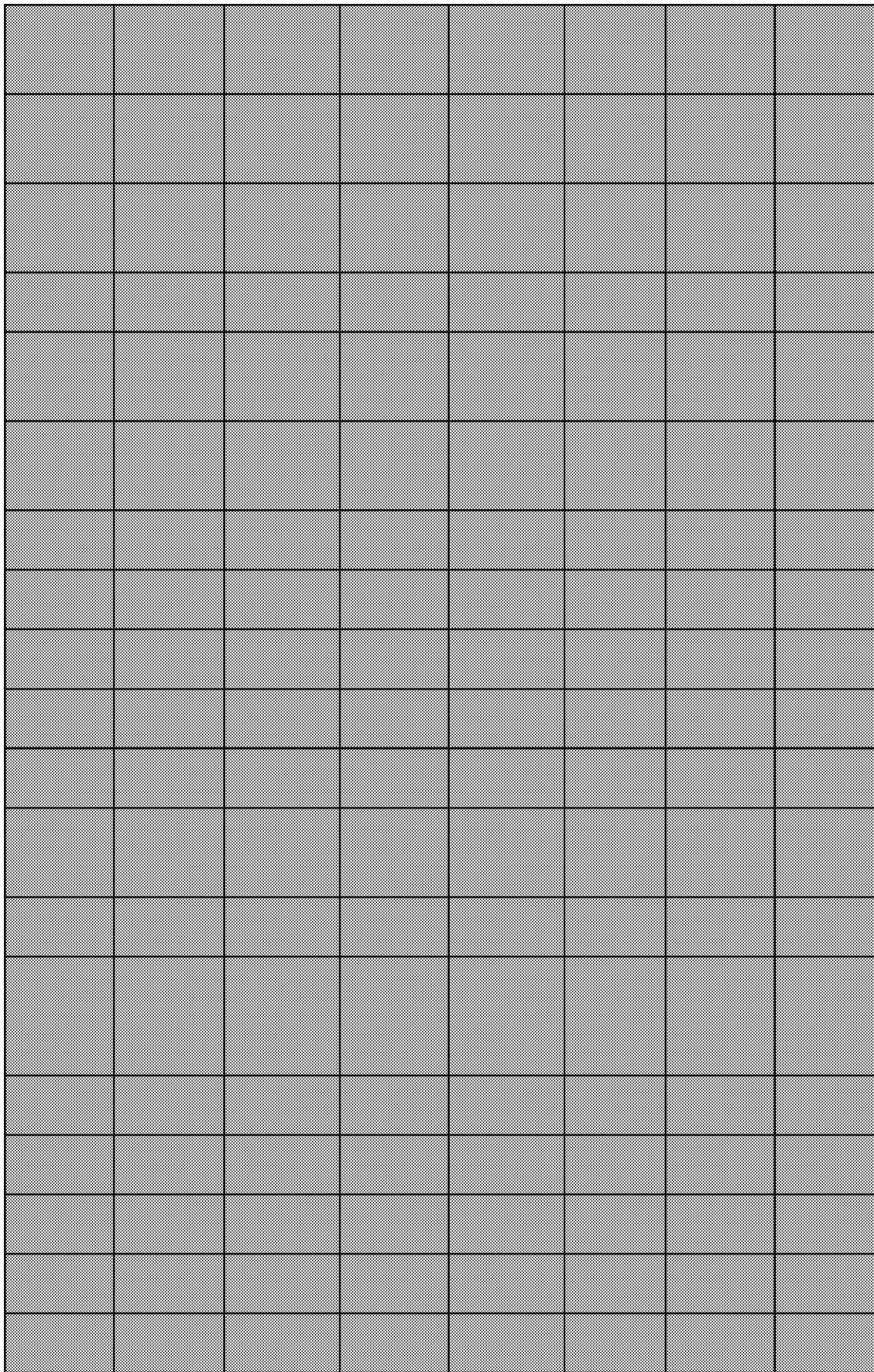


Level 1



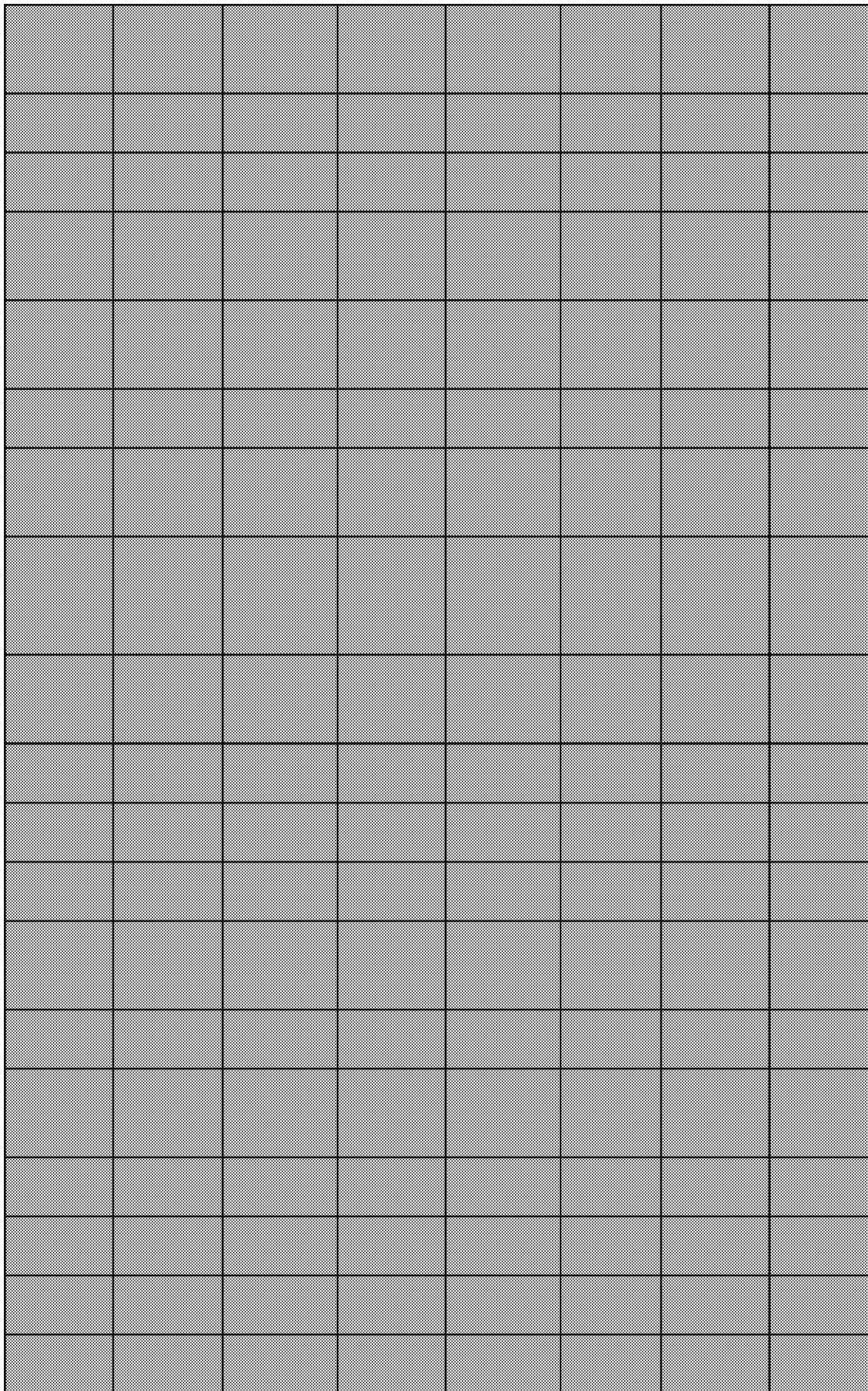
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In order to study oxidative stress in the lung, we have developed a rat lung slice model with compromised oxidative defense.
Dimethylthiourea (DMTU) is an effective scavenger of reactive oxygen metabolites. This property has been successfully exploited to protect against paraquat-induced lung damage.
The in vivo effect of melatonin on paraquat-induced oxidative damage in rat lung and liver was studied using two parameters.
OBJECTIVES: Paraquat (PQ) is a widely used herbicide. Exposure to PQ at toxic doses can result in fatal acute lung injury. The widespread use of the nonselective contact herbicide paraquat (PQ) has been the cause of thousands of deaths from acute lung injury.
Peroxiredoxin 6 (Prdx6), a bifunctional 25-kDa protein with both GSH peroxidase and phospholipase A2 activities, is the critical enzyme involved in the protection of the lung from PQ-induced damage.
Mice with knock-out of peroxiredoxin 6 (Prdx6), a recently described antioxidant enzyme, were evaluated for susceptibility to PQ-induced lung damage.
Oxidative stress plays a prominent role in the pathophysiology of cystic fibrosis (CF). Despite the presence of oxidative stress, the lung of CF patients is relatively resistant to PQ-induced damage.
The effects of perfluorocarbon (PFC) on paraquat (PQ) induced acute lung injury (ALI) was evaluated among rats. Twenty rats were divided into four groups of five rats each.
In 14 beagle dogs, paraquat was infused in fractional doses to produce pulmonary fibrosis while avoiding fatal liver and kidney damage.
In the present investigation a possible involvement of tachykinins during sulfur dioxide-(SO ₂) and metabisulfite-(MBS) induced lung damage was examined.
1. 3H-Dopa is converted by lung and liver microsomes to a reactive intermediate which binds covalently to lung and liver proteins.
The herbicide paraquat causes lung injury that is believed to be oxygen-radical mediated. To further characterize this injury, the role of tachykinins was examined.
Estrogen protects females against cardiovascular diseases in both receptor-dependent, genomic or non-genomic manner.
Increased formation of reactive oxygen species is a cause of paraquat (PQ)-induced injury and also provides a link between estrogen and paraquat-induced lung damage.
Paraquat, one of the most widely used herbicides, is highly toxic to humans and animals. There is much information regarding its mechanism of action.
The ability of melatonin to protect against paraquat-induced oxidative damage in rat lung, liver, and serum was examined.
The pharmacokinetics of paraquat were examined at a dose which produced lung disease but avoided renal damage. Following oral administration, paraquat was absorbed rapidly from the gut.
The paper presents results showing differential response to paraquat toxicity in Wistar rats and Swiss strain of mice. Paraquat is a potent inducer of oxidative stress in the lung.

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The high Paraquat (PQ, 1-1'-dimethyl-4,4'bipyridinium dichloride) embryotoxicity in *Xenopus laevis* has been shown to be

Rats exposed to the quaternary herbicide paraquat (PQ) exhibit oxidative stress and lung injury. In the present study, we

Paraquat (PQ) overdose can cause acute lung injury and death. Ozone therapy (OT) was previously demonstrated to alleviate

BACKGROUND: Mineralocorticoid receptor (MR) antagonists attenuate renal injury in salt-sensitive hypertensive rats without

OBJECTIVE: to study the oxidative stress of rats with acute paraquat poisoning and the intervention of Sodium Dimercaptosuccinate (SDS)

In order to facilitate the study of oxidative stress in lung tissue, rat lung slices with impaired antioxidant defenses were prepared.

Although paraquat (PQ) is widely known to induce pulmonary fibrosis, the molecular mechanisms are poorly understood.

Several mechanisms leading to noncardiac pulmonary edema have been reviewed. Common features are damage to alveolar capillary barrier and increased vascular permeability.

The metabolism of paraquat generates oxygen radicals. Paraquat has thus been suggested as a model compound to induce oxidative stress.

Shaking behavior, so-called wet dog shakes (WDS), in rats is characteristic behavior indicating morphine abstinence in most cases.

In both paraquat and X/XO models of lung injury, the injury, previously attributed to the generation of reactive oxygen species (ROS),

At low concentrations, nitric oxide is a physiological transmitter, but in excessive concentrations it may cause cell and tissue damage.

We investigated the effects of N-acetylcysteine (NAC) pretreatment on paraquat-induced lung inflammation and leak. We found that NAC reduced the leakage of albumin from the lungs.

We investigated a possible role for N-acetylcysteine (NAC), a well-known antioxidant and free radical scavenger, against paraquat-induced lung injury.

We have reviewed some of the factors which contribute to lung damage by various toxicants. These include disposition of toxicants, their metabolic activation, and cellular responses.

The primary ultrastructural changes in pulmonary alveolar epithelial cells are described in paraquat-injected rats. Within 24 h after injection, the alveolar epithelial cells show degenerative changes.

OBJECTIVE: To investigate the effects of overexpression of nuclear factor E2-related factor-2 (NRF2) on lung injury in rats.

Methylene blue (MB) attenuates acute lung injury (ALI) induced by liver or pancreas ischemia-reperfusion. But the protective mechanism of MB is not clear.

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